Spring 2017

Identifying Kinase Genes Involved in Regulation of the Stem Cell versus Meiotic Fate Decision in the C. elegans Germline

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Germline stem cells make a decision between maintaining the stem cell fate or entering meiosis. In the nematode *Caenorhabditis elegans*, germ cells adjacent to the somatic niche, the distal tip cell (DTC) of the adult germline, maintain the stem cell fate through the action of the GLP-1 Notch signaling pathway, while germ cells away from the influence of the DTC enter meiosis. *glp-1* maintains the stem cell fate through repression of the GLD-1 and GLD-2 pathways, which function redundantly to promote meiotic entry. The mechanism whereby *glp-1* signaling represses the GLD-1 and GLD-2 pathways is not completely understood, although it is known that regulation of the GLD-1 and GLD-2 pathways is post-transcriptional. I postulated that individual kinase genes, which act post-translationally, may function to promote meiotic entry in the *C. elegans* germline by promoting GLD-1 and/or GLD-2 pathway activity or inhibiting *glp-1* activity. With the exception of *glp-1*, due to genetic redundancy, loss of function of known single genes does not have a significant effect on the stem cell versus meiotic fate decision. Therefore, I used a sensitized genetic background, *glp-1*(*ar202*), to identify kinases, following RNAi knockdown, that result in a tumorous germline due to failure to switch to the meiotic fate. RNAi of 276 *C. elegans* kinase genes identified 26 that show incompletely penetrant germline tumor phenotypes. A deletion mutant for one kinase, *unc-82*, crossed into *glp-1*(*ar202*) background, significantly increased germline overproliferation, confirming that *unc-82* acts to promote GLD-1 and/or GLD-2 activity or inhibit *glp-1*. Future experiments will determine if deletion mutants of any of the remaining kinase genes results in the formation of germline tumors.